



Radiographic Presentations of Hospital Acquired Pneumonia in Pediatric ICU

**Nahla Mohamed Heikal^{1*}, Mohamed Adel Eltomey²,
Sahar Mohey El-Din Hazzaa³ and Khaled Talaat Muhammad¹**

¹*Pediatric Department, Faculty of Medicine, Tanta University, Egypt.*

²*Radiological Department, Faculty of Medicine, Tanta University, Egypt.*

³*Biochemistry Department, Faculty of Medicine, Tanta University, Egypt.*

Authors' contributions

This work was carried out in collaboration among all authors. Author NMH designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors KTM, MAE and SMEDH managed the analyses of the study. Author KTM managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

Background: Hospital-acquired pneumonia is a major medical problem even in developed countries. It is the most common nosocomial infection reaching 25% of all infections in the intensive care unit (ICU).

Aim: Aim is to study the radiographic findings of hospital acquired pneumonia in collaboration with laboratory and clinical findings in pediatric intensive care unit.

Patients and Methods: A prospective study on 60 pediatric patients admitted to PICU. Cases were divided into two groups. Group A: 30 cases with clear chest x-ray on admission and developed Hospital Acquired Pneumonia (HAP) after 48 hours. Group B: 30 cases with Community Acquired Pneumonia (CAP) on admission. Both groups were subdivided into mechanically ventilated and non-Mechanically Ventilated (MV and non-MV).

Results: Regarding X-ray in 1st day there was significant increase in CAP compared with HAP in the form of Bronchopneumonia and lobar pneumonia with effusion. X-ray in HAP had significant

*Corresponding author: E-mail: nahlaheikal92@gmail.com;

worsening in 3rd day compared with 1st day in both MV and non-MV groups. Otherwise no difference was found between groups.

Regarding CT Chest, there was statistically significant increase in Bronchopneumonia in non-MV CAP compared with other groups. Also, there was statistically significant increase in Rt. Upper lobar pneumonia in MV HAP compared with other groups. Similarly, there was statistically significant increase in Lt. pleural effusion with underlying consolidation collapse of lower lobe in MV HAP compared with other groups. There was statistically significant increase in Rt. pleural effusion with underlying consolidation collapse of rt. Lung in non-MV CAP compared with other groups. There was statistically significant increase in Bronchopneumonia with Rt. minimal pneumothorax in MV CAP compared with other groups. Otherwise, there was no significant difference between the studied groups.

Conclusion: Hospital acquired pneumonia was worse radiologically and bacteriologically. Hence, need more time to heal and more aggressive therapy was needed. Clinical pulmonary infection score was predictor for mortality. Predictors for length of stay (LOS) were found total leukocystic count (TLC), Absolute Neutrophilic Count (ANC), ESR and Culture & Sensitivity of bronchial secretions.

Keywords: Radiography; hospital acquired; pneumonia; pediatric; ICU.

1. INTRODUCTION

Several reports published confirm that hospital-acquired pneumonia (HAP) remains to be a major medical problem in most European countries and in the United States despite the advances in the quality of patient care, availability of effective antibiotics, complex technological diagnostic facilities and awareness in infection control measures [1].

Hospital-acquired pneumonia is considered one of the most common nosocomial infections which accounts for approximately 25% of all infections in the intensive care unit (ICU) [2].

Its occurrence represents additional cost, morbidity and most importantly, mortality among patients hospitalized initially for other reasons. The reported frequency varies with the definition, type of ICU, patients' population, and antibiotic policies [3].

Etiologic diagnosis of HAP is considered a microbiological emergency because of its impact on disease associated morbidity and mortality and antibiotic management. So, rapid diagnostic information is clearly more beneficial to patients than more complete but delayed information [4].

While, HAP is closely related to ventilator-associated pneumonia (VAP) that refers to pneumonia that arises more than 48–72 hours after endotracheal intubation and the cause of infection is usually multi-drug resistant (MDR) bacteria [5].

There are many risk factors associated with HAP, and VAP including many environmental and pharmacological factors [6].

The diagnosis of HAP is mainly clinical, through the endotracheal aspirate (ETA) cultures, white blood cell (WBC) count, serial chest radiographs and arterial blood gases (ABG) [7].

The value of radiological examination on admission and later during the PICU stay was needed to be evaluated as a reliable method of diagnosis.

2. MATERIALS AND METHODS

A prospective study on 60 pediatric patients from 3 to 168 months (36 males, 24 females) admitted to PICU. Tanta University Hospital from December 2018 to December 2019.

Cases were divided into two groups. Each group was divided into ventilated and non-ventilated subgroups.

Group A: Thirty cases with clear chest x-ray on admission and developed Hospital Acquired Pneumonia after 48 hours (MV 18, Non-MV 12).

Group B: Thirty cases with Community Acquired Pneumonia on admission (MV 10, Non-MV 20).

Inclusion criteria were patients admitted to PICU with pneumonia either Hospital acquired, or Community acquired pneumonia.

The exclusion criteria were: Patient with brain death, Congenital pulmonary diseases, Associated disease (e.g.: heart failure, acute kidney injury etc.), Admission to other hospital before Tanta University PICU, or Receiving antibiotic treatment before admission.

2.1 Statistical Analysis

Data were computed and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp) Qualitative data were described using number and percent. Chi-square test was used for categorical variables, to compare between different groups. Wilcoxon signed ranks test for abnormally distributed quantitative variables, to compare between two periods. Student t-test for normally distributed quantitative variables, to compare between two studied groups. Mann Whitney test for abnormally distributed quantitative variables, to compare between two studied groups.

Methods Clinical history and examination, Laboratory investigations and CT chest when possible were done. All the patients were monitored for Oxygen saturation, systolic and diastolic blood pressure, heart rate and RR. Radiological investigations included Chest X-ray. Transcutaneous blood gases, Clinical Pulmonary Infection Score (CPIS) and Oxygenation index (OI) were measure.

3. RESULTS

Regarding demographic data of the studied groups there was statistically significant decrease in the age of MV HAP compared with other groups. Otherwise, there was no significant difference between the studied groups (Table 1).

Regarding Temperature there was statistically significant increase in non-MV HAP compared with MV HAP. Otherwise, there was no significant difference between the studied groups. Regarding Systolic Blood Pressure There was statistically significant decrease in MV HAP compared with Non-MV HAP. Also, there was statistically significant decrease in MV HAP compared with Non-MV CAP. Regarding Diastolic Blood Pressure There was statistically significant decrease in MV HAP compared with other groups. Otherwise, there was no significant difference between the studied groups (Table 2).

Regarding TLC in 1st day there was significant increase in MV CAP compared with MV and

Non-MV HAP. Also, significant increase in Non-MV CAP compared with MV and Non-MV HAP. In 3rd day there was significant increase in MV HAP compared with MV and Non-MV CAP, also significant increase in Non-MV HAP compared with MV and Non-MV CAP. Regarding TLC in CAP there was significant increase in 1st day compared with 3rd day in both groups MV and non-MV. Regarding TLC in HAP there was significant increase in 3rd day compared with 1st day in both groups MV and non-MV. Otherwise, there was no significant difference between the studied groups. Regarding ANC, in 1st day there was significant increase in MV CAP compared with MV and Non-MV HAP, also significant increase in Non-MV CAP compared with MV and Non-MV HAP. In 3rd day there was significant increase in MV HAP compared with MV and Non-MV CAP, also significant increase in Non-MV HAP compared with MV and Non-MV CAP. Regarding ANC in CAP there was significant increase in 1st day compared with 3rd day in both groups MV and non-MV. Regarding ANC in HAP there was significant increase in 3rd day compared with 1st day in both groups MV and non-MV. Otherwise, there was no significant difference between the studied groups (Table 3).

CRP in 1st day there was significant increase in MV CAP compared with MV and Non-MV HAP. Also, significant increase in Non-MV CAP compared with MV and Non-MV HAP. Regarding 3rd day there was significant increase in MV HAP compared with MV and Non-MV CAP, also significant increase in Non-MV HAP compared with MV and Non-MV CAP. Regarding CRP in CAP there was significant increase in 1st day compared with 3rd day in both groups MV and non-MV. Regarding CRP in HAP there was significant increase in 3rd day compared with 1st day in both groups MV and non-MV. Otherwise, there was no significant difference between the studied groups. ESR in 1st day there was significant increase in MV CAP compared with MV and Non-MV HAP, also significant increase in Non-MV CAP compared with MV and Non-MV HAP. Regarding 3rd day there was significant increase in MV HAP compared with MV and Non-MV CAP, also significant increase in Non-MV HAP compared with MV and Non-MV CAP. Regarding ESR in CAP there was significant increase in 1st day compared with 3rd day in both groups MV and non-MV. Regarding ESR in HAP there was significant increase in 3rd day compared with 1st day in both groups MV and non-MV. Otherwise, there was no significant difference between the studied groups (Table 4).

Table 1. Demographic data of the studied groups

	HAP				CAP				Test of Sig.	p
	MV (n= 18)		Non MV (n= 12)		MV (n= 10)		Non MV (n= 20)			
	No.	%	No.	%	No.	%	No.	%		
Sex										
Male	10	55.6	8	66.7	5	50.0	13	65.0	$\chi^2=$	^{MC} p=
Female	8	44.4	4	33.3	5	50.0	7	35.0		
Age (months)										
Min. – Max.	3.0 – 144.0		4.0 – 98.0		4.0 – 168.0		5.0 – 132.0		H=	0.007*
Median	4.50		39.0		60.0		35.0		12.143*	
Sig. bet. grps.	p ₁ =0.039*, p ₂ =0.006*, p ₃ =0.002*, p ₄ =0.465, p ₅ =0.519, p ₆ =0.842									

CAP: Community acquired pneumonia, HAP: Hospital acquired pneumonia, MV: Mechanical ventilation, Non MV: Not on mechanical ventilation

Table 2. Vital data of the studied groups

Vital data	HAP		CAP		F	P
	MV (n = 18)	Non MV (n = 12)	MV (n = 10)	Non MV (n = 20)		
Temp (°C)						
Min. – Max.	36.80 – 40.0	38.70 – 40.0	37.90 – 39.50	37.90 – 39.40	4.077	0.011*
Median	38.70	39.0	38.65	38.95		
RR (breath/min)						
Min. – Max.	38.0 – 55.0	35.0 – 55.0	35.0 – 50.0	35.0 – 56.0	0.114	0.952
Median	47.0	48.0	47.0	45.50		
HR (beat/min.)						
Min. – Max.	128.0 – 165.0	120.0 – 165.0	110.0 – 155.0	115.0 – 160.0	1.469	0.233
Median	142.0	145.0	135.0	136.0		
Systolic blood pressure (mmHg)						
Min. – Max.	65.0 – 125.0	90.0 – 125.0	90.0 – 110.0	90.0 – 110.0	4.704*	0.005*
Median	82.50	100.0	100.0	100.0		
Diastolic blood pressure (mmHg)						
Min. – Max.	35.0 – 70.0	65.0 – 80.0	55.0 – 75.0	60.0 – 75.0	15.044*	<0.001*
Median	52.50	70.0	65.0	70.0		

RR: Respiratory rate, Temp.: Temperature, HR: Heart rate

C&S of Bronchial Secretions showed a statistically significant increase in (Klebsiella and Pseudomonas) in MV HAP compared with other studied groups. There was statistically significant increase in MRSA in Non-MV HAP compared with other studied groups. There was statistically significant increase in Acinetobacter in HAP compared with CAP. Also, there was statistically significant increase in (Strept. pneumoniae and fungal) in CAP compared with HAP. Otherwise, there was no significant difference between the studied groups (Table 5 and Figs. 1-5).

Regarding X-ray in 1st day there was significant increase in CAP compared with HAP in the form of Bronchopneumonia and lobar pneumonia with effusion. Regarding X-ray in HAP there was significant worsening in 3rd day compared with 1st

day in both MV and non-MV groups. Otherwise, there was no significant difference between the studied groups (Table 6 and Fig. 6).

Regarding CT Chest, there was statistically significant increase in Bronchopneumonia in non-MV CAP compared with other groups. Also, there is statistically significant increase in Rt. Upper lobar pneumonia in MV HAP compared with other groups. Similarly, there was statistically significant increase in Lt. pleural effusion with underlying consolidation collapse of lower lobe in MV HAP compared with other groups. There was statistically significant increase in Rt. pleural effusion with underlying consolidation collapse of rt. Lung in non-MV CAP compared with other groups. There was statistically significant increase in Bronchopneumonia with Rt. minimal pneumothorax in MV CAP compared with other

groups. Otherwise, there was no significant difference between the studied groups (Table 7).

Regarding Oxygenation Index, there was no statistically significant difference between studied groups (Table 8).

Regarding CPIS Score, there was statistically significant increase in MV HAP compared with MV CAP. Otherwise, there was no significant difference between the studied groups (Table 9).

Table 3. Total leucocytic count (x10³/mm³) and absolute neutrophilic count (x10³/ mm³) of the studied groups

	HAP		CAP		F	P
	MV (n = 18)	Non MV (n = 12)	MV (n = 10)	Non MV (n = 20)		
TLC(x10³/mm³)						
1st day						
Min. – Max.	4.30 – 11.90	4.80 – 8.70	15.40 – 22.80	14.70 – 21.60	113.696*	<0.001*
Median	8.10	6.60	16.40	16.50		
3rd day						
Min.– Max.	16.40 – 23.20	15.70 – 23.50	10.30 – 13.60	10.40 – 14.60	72.687*	<0.001*
Median	18.55	18.30	11.85	12.0		
p ₇	<0.001*	<0.001*	<0.001*	<0.001*		
ANC(x10³/ mm³)						
1st day						
Min. – Max.	2.30 – 7.79	2.90 – 4.90	11.60 – 18.30	11.40 – 18.90	117.233*	<0.001*
Median	4.75	4.0	12.70	12.45		
3rd day						
Min. – Max.	12.90 – 18.50	12.80 – 21.30	7.40 – 9.50	7.80 – 10.80	90.250*	<0.001*
Median	15.40	15.50	8.70	9.20		
p ₇	<0.001*	<0.001*	<0.001*	<0.001*		

ANC: Absolute Neutrophilic count, TLC: Total Leucocytic count

Table 4. C-reactive protein (mg/L) and erythrocyte sedimentation rate (mm) of the studied groups

	HAP		CAP		H	P
	MV (n = 18)	Non MV (n = 12)	MV (n = 10)	Non MV (n = 20)		
CRP (mg/L)						
1st day						
Min. – Max.	3.0 – 50.0	6.0 – 12.0	33.0 – 88.0	33.0 – 98.0	44.151*	<0.001*
Median	6.0	6.0	37.0	56.0		
3rd day						
Min. – Max.	12.0 – 88.0	22.0 – 48.0	12.0 – 28.0	19.0 – 33.0	12.611*	0.006*
Median	27.0	34.0	21.0	24.0		
p ₇	<0.001*	0.002	0.005*	<0.001*		
ESR						
1st day						
Min. – Max.	11.0 – 23.0	11.0 – 15.0	32.0 – 43.0	30.0 – 45.0	204.204*	<0.001*
Median	12.0	12.0	35.0	37.50		
3rd day						
Min. – Max.	16.0 – 36.0	18.0 – 26.0	18.0 – 22.0	17.0 – 22.0	4.618*	0.006*
Median	22.50	21.50	19.50	19.0		
p ₇	<0.001*	<0.001*	<0.001*	<0.001*		

CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate

Table 5. Culture and sensitivity of bronchial secretions of the studied groups

C&S of Bronchial secretions	HAP				CAP				MC p
	MV (n = 18)		Non MV (n = 12)		MV (n = 20)		Non MV (n = 30)		
	No.	%	No.	%	No.	%	No.	%	
No growth	0	0.0	0	0.0	4	40.0	8	40.0	<0.001*
Klebsiella	11	61.1	6	50.0	0	0.0	0	0.0	
Fungal	0	0.0	0	0.0	0	0.0	2	10.0	
MRSA	2	11.1	3	25.0	0	0.0	0	0.0	
Staph aureus	0	0.0	0	0.0	2	20.0	4	20.0	
Acenitobacter	2	11.1	2	16.7	0	0.0	0	0.0	
Pseudomonas	3	16.7	1	8.3	0	0.0	0	0.0	
Strept pneumoniae	0	0.0	0	0.0	4	40.0	6	30.0	

C&S: Culture and sensitivity

Table 6. X-Ray of the studied groups

X- Ray	HAP				CAP				MC p
	MV (n = 18)		Non MV (n = 12)		MV (n = 10)		Non MV (n = 20)		
	No.	%	No.	%	No.	%	No.	%	
1st day									
Clear	18	100.0	12	100.0	0	0.0	0	0.0	<0.001*
Bronchopneumonia	0	0.0	0	0.0	8	80.0	15	75.0	
Lobar pneumonia with effusion	0	0.0	0	0.0	2	20.0	5	25.0	
Lobar pneumonia	0	0.0	0	0.0	0	0.0	0	0.0	
3rd day									
Clear	0	0.0	0	0.0	0	0.0	0	0.0	0.090
Bronchopneumonia	9	50.0	4	33.3	8	80.0	15	75.0	
Lobar pneumonia with effusion	3	16.7	3	25.0	2	20.0	3	15.0	
Lobar pneumonia	6	33.3	5	41.7	0	0.0	2	10.0	
MR p₁	<0.001*		0.001*		1.000		0.157		

Table 7. CT chest of the studied groups

CT chest	HAP				CAP				MC p
	MV (n = 18)		Non MV (n = 12)		MV (n = 10)		Non MV (n = 20)		
	No.	%	No.	%	No.	%	No.	%	
Bilateral pneumonia	6	40.0	3	27.3	5	62.5	11	68.8	0.038*
Rt. upper lobar pneumonia	5	33.3	3	27.3	0	0.0	0	0.0	
Lt. pleural effusion with underling consolidation collapse of lower lobe	3	20.0	1	9.1	1	12.5	1	6.3	
Rt. pleural effusion with underling consolidation collapse of rt. Lung	1	6.7	2	18.2	1	12.5	4	25.0	
Lt. lower lobar pneumonia	0	0.0	2	18.2	0	0.0	0	0.0	
Bilateral pneumonia with Rt. minimal pneumothorax	0	0.0	0	0.0	1	12.5	0	0.0	

Table 8. Oxygenation index of the studied groups

OI	MV HAP (n = 18)	MV CAP (n = 10)	T	p
Min. – Max.	2.0 – 19.0	2.0 – 19.0		0.114
Median	8.0	8.5		0.910

OI: Oxygenation index

Regarding length of stay, there was statistically significant increase in MV HAP compared with MV and Non-MV CAP. There was statistically significant increase in Non-MV HAP compared with MV and Non-MV CAP. Otherwise, there was no significant difference between the studied groups (Table 10).

Univariate and Multivariate predicting mortality showed that CPIS was significant in predicting mortality (Table 11).

Univariate and Multivariate for affecting of the LOS > 7 days show that TLC, ANC, ESR (1st

day), C&S of bronchial secretions was significant as a Univariate, but nothing was significant as Multivariate (Table 12).

4. DISCUSSION

Pneumonia is one of the most common infections in the PICU. This infection encompasses two different entities: VAP and HAP. The incidence of VAP ranges from 1.9 to 3.8 per 1000 days of mechanical ventilation in the US and exceeds 18 per 1000 days of mechanical ventilation in Europe [8].

Table 9. Clinical pulmonary infection score of the studied groups

CPIS score	HAP		CAP		H	P
	MV (n = 18)	Non MV (n = 12)	MV (n = 10)	Non MV (n = 20)		
Min. – Max.	2.0 – 9.0	3.0 – 10.0	1.0 – 7.0	3.0 – 7.0	8.792	0.032
Median	5.0	5.0	4.0	4.0		
Sig. bet. grps.	p ₁ =0.318, p ₂ =0.004, p ₃ =0.066, p ₄ =0.411, p ₅ =0.131, p ₆ =0.607					

CPIS: Clinical pulmonary infection score

Table 10. Length of stay of the studied groups

Duration of hospitalization	HAP		CAP		H	P
	MV (n = 18)	Non MV (n = 12)	MV (n = 10)	Non MV (n = 20)		
Min. – Max.	18.0 – 90.0	18.0 – 45.0	7.0 – 16.0	6.0 – 22.0	41.818	<0.001
Median	35.0	25.50	8.50	9.0		
Sig. bet. grps.	p ₁ =0.484, p ₂ <0.001, p ₃ <0.001, p ₄ <0.001, p ₅ <0.001, p ₆ =0.678					

Table 11. Univariate and multivariate analysis for the parameters affecting for mortality for total sample

	Univariate		*Multivariate	
	P	OR (95%C.I)	p	OR (95%C.I)
Sex (male)	0.898	1.086(0.308 – 3.828)		
Age	0.338	0.992(0.975 – 1.009)		
Temp	0.334	0.620(0.235 – 1.635)		
RR	0.797	0.986(0.884 – 1.100)		
HR	0.432	1.019(0.972 – 1.069)		
Systolic blood pressure (mmHg)	0.711	0.991(0.946 – 1.039)		
Diastolic blood pressure (mmHg)	0.330	0.973(0.921 – 1.028)		
Duration of hospitalization (>7)	0.417	2.462(0.279 – 21.715)		
X- Ray (Bronchopneumonia +localized)	0.754	0.821(0.240 – 2.814)		
TLC (x10 ³) (1 st day)	0.847	1.011(0.902 – 1.134)		
ANC (x10 ³) (1 st day)	0.851	0.988(0.871 – 1.120)		
CRP (1 st day)	0.924	1.001(0.979 – 1.024)		
ESR (1 st day)	0.676	1.011(0.960 – 1.065)		
C&S of Bronchial secretions Score (1st day)	0.001	2.273(1.426 – 3.624)	0.001*	2.273(1.426 – 3.624)
HAP	0.870	1.112(0.309 – 4.0)		
MV	0.603	1.404(0.391 – 5.043)		

Table 12. Univariate and multivariate analysis for the parameters affecting for LOS (>7 days) for total sample

	Univariate		#Multivariate	
	P	OR (95%C.I.)	p	OR (95%C.I.)
Sex (male)	0.251	0.377(0.071 – 1.993)		
Age	0.910	0.999(0.983 – 1.016)		
Temp	0.607	0.715(0.199 – 2.564)		
RR	0.910	0.993(0.874 – 1.127)		
HR	0.253	0.967(0.913 – 1.024)		
Systolic blood pressure (mmHg)	0.281	0.968(0.913 – 1.027)		
Diastolic blood pressure (mmHg)	0.216	0.938(0.848 – 1.038)		
X- Ray (Bronchopneumonia +localized)	0.998	–		
TLC (x10 ³) (1 st day)	0.046*	0.855(0.733 – 0.977)	0.606	1.489(0.328 – 6.752)
ANC (x10 ³) (1 st day)	0.042*	0.847(0.721 – 0.994)	0.849	0.869(0.205 – 3.680)
CRP (1 st day)	0.072	0.977(0.953 – 1.002)		
ESR (1 st day)	0.017*	0.891(0.811 – 0.980)	0.106	0.811(0.629 – 1.046)
C&S of Bronchial secretions Score (1 st day)	0.001*	15.0(2.948 – 76.310)	0.063	5.460(0.914 – 32.607)
HAP	0.998	–		
MV	0.390	1.923(0.433 – 8.539)		

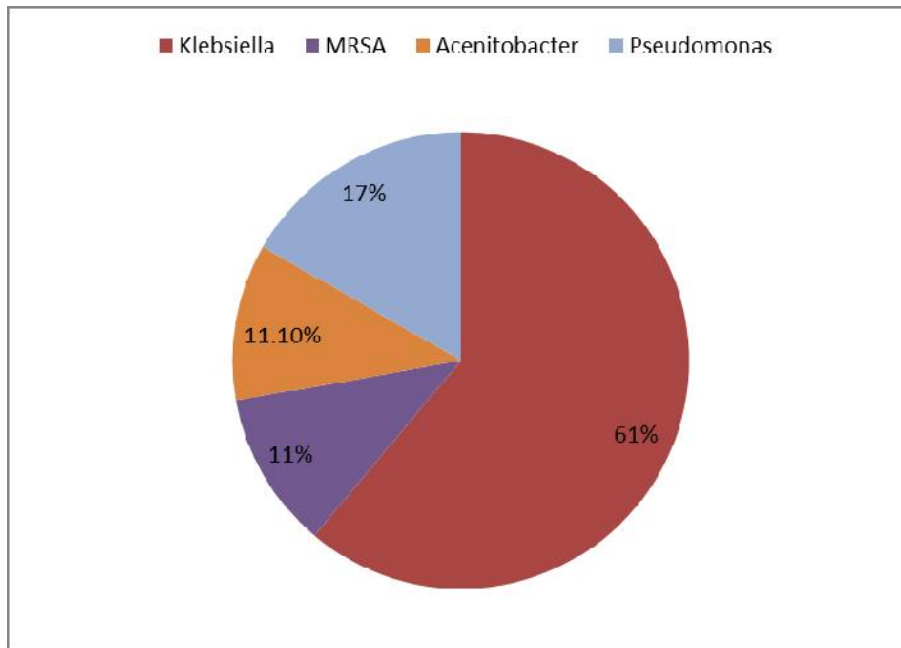


Fig. 1. C&S of bronchial secretions of MV HAP

Nosocomial pneumonia is the most common infection in PICU, when considering the timing of these infections. Non-MV HAP occurs in patients admitted to the hospital for at least 48 hours and VAP is defined as occurring more than 48 hours after the initiation of mechanical ventilation.

Accurate data on their epidemiology are limited by the lack of standardized diagnostic criteria. In the US, the incidence of non-ventilator-HAP was 1.6%, representing a rate of 3.63 per 1000 patient-days. Hospital acquired pneumonia in the PICU is associated with an approximate mortality

rate of 20%. Diagnosis relies on clinical assessment and microbiological findings [9].

Community-acquired pneumonia is the leading cause of childhood morbidity and mortality and responsible for approximately 1.4 million deaths per year which represents 18.3% of all deaths in children < 5 years [10].

Many microorganisms can cause childhood pneumonia both bacterial and viral and sometimes it is caused by multiple pathogens at once as a co-infection [11]. Although the direct impact of this infection on mortality remains debated, it is nonetheless associated with increased morbidity through increased duration of mechanical ventilation (or decrease in ventilator-free days) and increased PICU and hospital Length of Stay (LOS) [8].

The goal of this work was to study the radiographic findings of hospital acquired pneumonia in collaboration with laboratory and clinical findings in pediatric intensive care unit. The present study was conducted over one year from December 2018 to December 2019 upon sixty critically ill children and infants admitted to Tanta university hospital, PICU who were divided into two groups, Group A thirty cases with clear CXR on admission and developed HAP after 48 hours, Group B thirty cases with CAP on admission, Each group was divided into MV and non-MV subgroups.

Chest X-ray was performed for all studied cases on admission then followed up after 48 hours. CT chest when it was possible (only 50 cases).

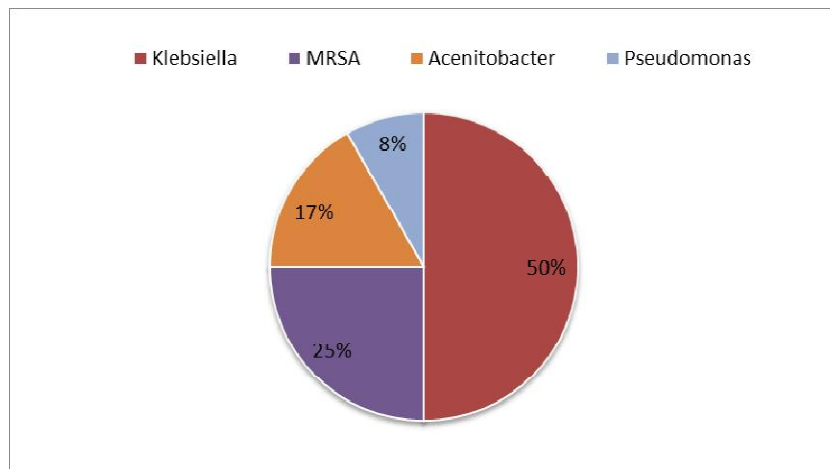


Fig. 2. C&S of bronchial secretions of Non-MV HAP

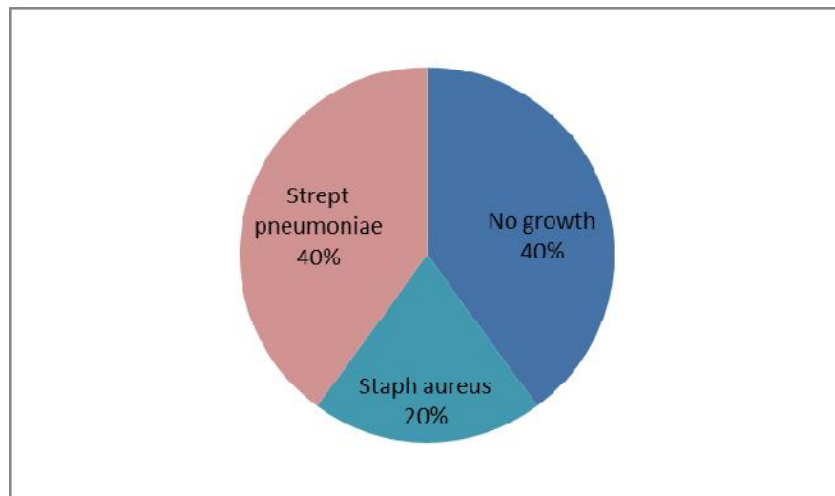


Fig. 3. C&S of Bronchial Secretions of MV CAP

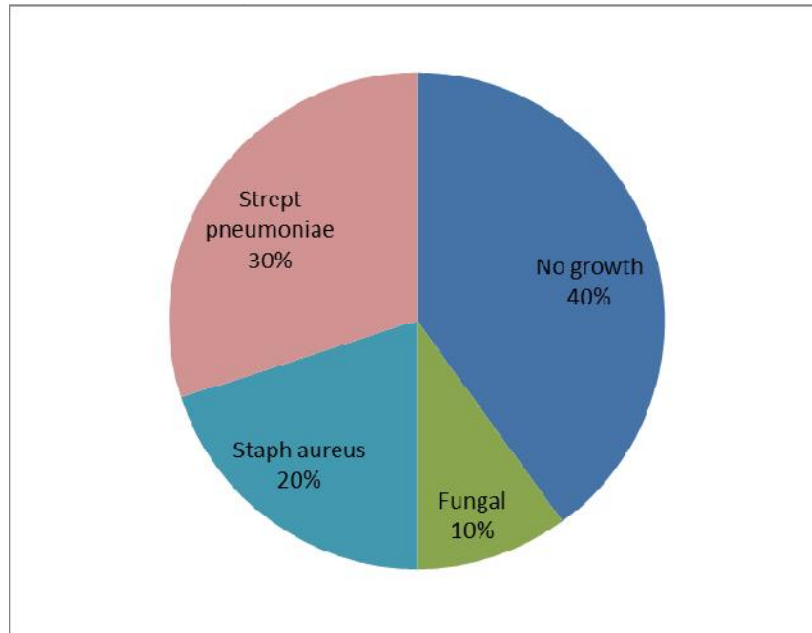


Fig. 4. C&S of bronchial secretions of Non-MV CAP

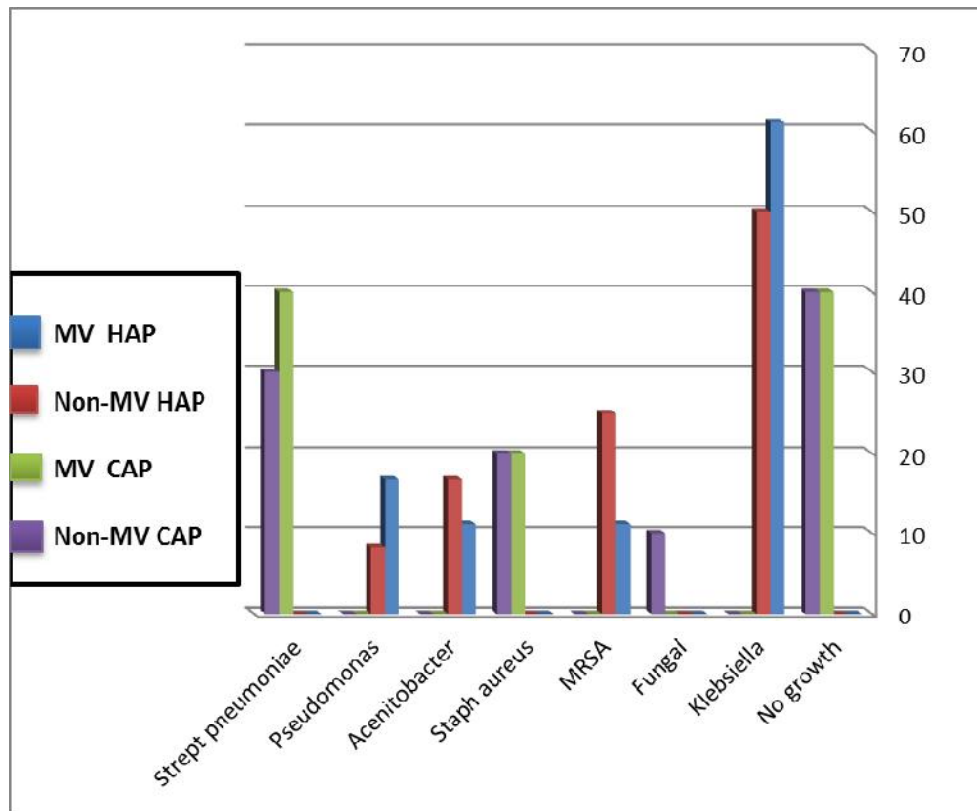


Fig. 5. C&S of bronchial secretions of the studied groups

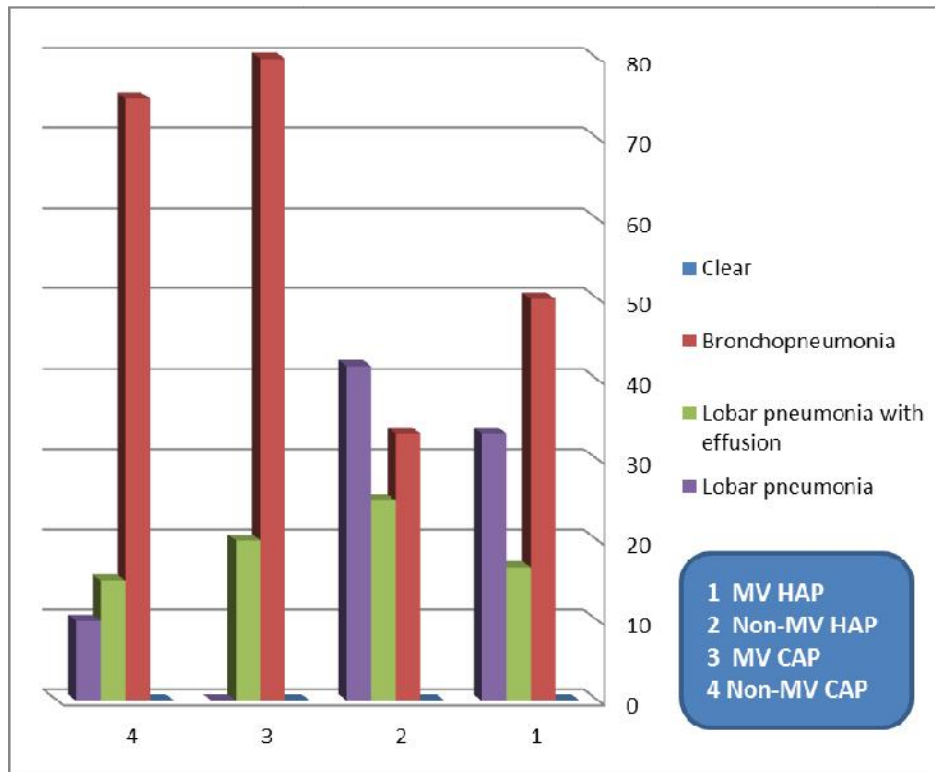


Fig. 6. CXR of the studied groups in the 3rd day

The study showed that there was decrease in the age of MV HAP compared with other groups.

This may be explained by that younger infants are more susceptible to be ventilated. Therefore, they have more risk to develop VAP.

This was in accordance with Gadappa et al. [12] who stated that (39.5%) patients were < 1year age. Likewise, large percentages are ventilated in infancy that makes them more liable for MV HAP.

Also, Liliana et al. [13] revealed that the most frequent MV HAP was among children less than 6 months. This study also showed that there was decrease in systolic blood pressure of MV HAP compared with non-MV HAP and Non-MV CAP and decrease in diastolic blood pressure of MV HAP compared with other groups.

This may be explained by that monitoring changes in heart rate and blood pressure can reflect an acute change in the patient's condition or trend impending problems. Ventilated patients frequently have an increase in heart rate to improve CO and to compensate decreased

oxygenation. Although, increasing heart rate is an effective mechanism for increasing the CO. There is a point when increasing the heart rate will cause the CO to decrease as the decreased time for ventricular filling which may affect blood pressure on mechanically ventilated patients, so it may be presented as decreased for other MV patients.

This study showed that TLC, ANC and ESR on admission increased in MV CAP compared with MV and Non-MV HAP. Also, increase in Non-MV CAP compared with MV and Non-MV HAP but in 3rd day follow up showed increase in MV HAP compared with MV and Non-MV CAP, also increase in Non-MV HAP compared with MV and Non-MV CAP. Also, in CAP there was increase in 1st day compared with 3rd day in both groups MV and non-MV and for HAP there was increase in 3rd day compared with 1st day in both groups MV and non-MV.

This was agreed with Russell et al. [14] radiologically confirmed HAP appears to be represented with significantly higher levels of inflammatory markers (white cell count, neutrophils, and C-reactive protein).

In this study organisms isolated from sputum culture of the studied cases was 17 cases (28.3%) were isolate *Klebsiella pneumoniae*, 4 cases (6.7%) were isolate *Pseudomonas aeruginosa*, 4 cases (6.7%) isolate *Acinetobacter* and 5 cases (8.3%) isolate MRSA.

Likewise, in 2015 In previous study in Tanta PICU Ibrahim, [15] showed that *Klebsiella pneumoniae* was the most frequently isolate 30.4%, followed by *Acinetobacter* 26.09%, coagulase negative staphylococci 13.04%, *S. aureus* 8.7%, *Pseudomonas aeruginosa* 8.7% and *Candida* species 8.7%.6.

Also, in El-Bayoumi et al. [16] A study in Mansoura University Pediatric Hospital enrolled children admitted to PICU for ≥ 48 hours, who acquired nosocomial infection. *Klebsiella* was the most common isolate (19.1%) followed by *Staphylococcus aureus* (12.2%), MRSA (6.5%).

Similar results were reported in Pediatric Department of the Santa Casa de São Paulo, Brazil, in Arnonie al. 2007 study, [17] the most prevalent agents were: *Klebsiella* 37%, *Acinetobacter baumannii* 21.7% then *Pseudomonas aeruginosa* 12%.

That was against with Gupta et al. In India [18] *Acinetobacter* species was the most common isolate organism 48% followed by *Pseudomonas aeruginosa* 32%, *Klebsiella* 23.6% and *Staphylococcus aureus* 10%.

An active surveillance program was implanted for 10 months in medical ICU for neonatal, pediatric and adults in 3 large tertiary care university hospitals in Egypt 2011 by ElKholy et al. [19] total of 600 pathogen were isolated from blood cultures of 1575 patients. The reported results showed that Gram-negative bacteria accounted for 61.7% of total pathogens. *Klebsiella* spp. Were the most common bacteria isolated 43.2%. Gram positive organisms constituted 34.5%.

On the other hand, Becerra et al. [20] reported that the most common isolate was *Pseudomonas aeruginosa* 29.8% then *Candida* spp. 28.3%, *Klebsiella* 8.9% and coagulase negative staphylococci 7.4%.

Klebsiella pneumoniae is a rare cause of CAP but accounts for a higher proportion of HAP, where patients are more likely to be treated with antibiotics that permit this bacterium to dominate the pharyngeal flora so make them susceptible

for aspiration making them most common organism for HAP [21].

This study revealed that CXR in HAP showed increased diffuse lung infiltrates, localized lobar consolidation with or without effusion in the after 48 hours follow up CXR compared with the on admission CXR in both groups MV and non-MV.

This was in harmony with Eida et al. [22] who found that plain posteroanterior CXR follow-up of the admitted patients to ICU; a newly evidence of developed pneumonia (as opacity of one lung segmental lobe, or bilateral opacities primarily in the bases of the lungs) was confirmed.

Similarly, Bendary et al. [23] showing CXR of cases with HAP presented with diffuse lung infiltrates or localized lobar consolidation.

This study also showed that about CT chest there is increase in Bronchopneumonia and Rt. pleural effusion with underling consolidation collapse of rt. Lung in non-MV CAP, increase in Bronchopneumonia with Rt. minimal pneumothorax in MV CAP, increase in Rt. Upper lobar pneumonia in MV HAP, increase in Lt. pleural effusion with underling consolidation collapse of lower lobe in MV HAP when compared with other groups.

This may be explained as CT chest useful in differentiating mimics from actual pneumonia and strength the diagnosis of pneumonia based on the clinical presence of fever, cyanosis, hypotension, and infiltrates (< 72 hours) on chest radiographs plus organismal growth in respiratory secretions.

Likely Nicolas et al. [24] concluded that CT-scan can improve the diagnosis and reclassification of patients with pneumonia. CT-scan is especially useful to rule-out pneumonia and has a maximal impact in the category of patients with intermediate probability of disease. This study showed increase in CPIS of MV HAP compared with other groups. The non-significant correlation of OI for differentiating MV HAP and MV CAP could be explained by the small number of cases. This study found that there is increase in CPIS for MV HAP compared with MV CAP.

This was in accordance with Luna et al. [25] shows that CPIS is significant in patients with MV HAP which enrolled 427 consecutive patients receiving mechanical ventilation in a prospective observational cohort study at 6 critical care units

in Argentina. Sixty-three patients were deemed to have MV HAP by both clinical and microbiologic criteria. The modified CPIS (microbiology data were excluded) was calculated both before and after the diagnosis of VAP. Although the CPIS increased consistently in all patients through the day of MV HAP diagnosis, it decreased significantly during the treatment phase in the survivors of MV HAP but remained elevated in the non survivors. This observation revealed that the CPIS correlated well with eventual mortality. However, not all components of the CPIS contributed equally to explaining outcome.

This study shows that regarding LOS there was increase in HAP compared with CAP. This was in accordance with Giuliano et al. [9] shows that HAP is responsible for prolonged LOS in hospitals.

This may be explained by the presence hap may add comorbidities to the admitted critically ill child to PICU that may prolong PICU stay through difficult mv weaning, reintubation or emerging respiratory distress. Univariate and multivariate predicted that CPIS was significant in predicting mortality and for affecting of the los more than 7 days showed that TLC, ANC, ESR and C&S of bronchial secretions was significant as a univariate predictors but nothing was significant as multivariate.

5. CONCLUSION

Hospital acquired pneumonia was worse radiologically and bacteriologically. Hence, need more time to heal and more aggressive therapy was needed. Clinical pulmonary infection score was predictor for mortality. Predictors for LOS were found TLC, ANC, ESR and C&S of bronchial secretions.

CONSENT AND ETHICAL APPROVAL

A written informed consent was obtained from the guardians of all patients included in the study. The study was approved by the Ethics committee of Faculty of Medicine, Tanta University.

Written informed consent will be obtained from the parents of all subjects of the study. All work will be in compliance with declaration of Helsinki. The study will be approved by the Ethics Committee of Faculty of Medicine, Tanta University. Permission number is 32651/10/18.

The waste product will be discarded according to the infection control policy of Tanta University Hospital.

Lowest dose of radiation to minimize the hazards of it for the children.

The risk to participants and measures used to minimize these risks:

When we take any sample we can introduce infection to the patient, and to minimize this risk the sample was taken under complete aseptic condition and was disposed according to the standard of infection control.

There is no other unexpected risks appeared during course of the research.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Dunn L. Pneumonia: Classification, diagnosis and nursing management. J. Nurs. Stand. 2005;19(42):50–54.
2. Bartlett JG. Hospital-acquired pneumonia: The Merck manual for healthcare professionals; 2008.
3. E Gu v MB, Voss A, Doganay M. Incidence, risk factors and mortality of nosocomial pneumonia in intensive care units: A prospective study. Ann Clin Microbiol Antimicrob. 2004;3:17.
4. Dodek P, Keenan S, Cook D, Heyland D, Jacka M, Hand L. For the Canadian Critical Care Trials Group and the Canadian Critical Care Society. Evidence-based clinical practice guideline for the prevention of ventilator-associated pneumonia. Ann Intern Med. 2004;141:305–13.
5. Cunha BA. Multi-drug resistant (MDR) *Klebsiella*, *Acinetobacter* and *Pseudomonas aeruginosa*. Antibiot. Clin. 2008;10:354–5.
6. Kollef MH. The prevention of ventilator-associated pneumonia. N. Engl. J. Med. 2009;340:627–34.
7. Bates JH, Wagers S, Lundblad L, et al. Nonlinearity of respiratory mechanics during bronchoconstriction with airway inflammation. J. Appl. Physiol. 2002;92(5):1802–7.

8. Koulenti D, Tsigou E, Rello J. Nosocomial pneumonia in 27 ICUs in Europe: Perspectives from the EU-VAP/CAP study. *Eur J Clin Microbiol Infect Dis.*; 2016.
9. Giuliano KK, Baker D, Quinn B. The epidemiology of non-ventilator hospital acquired pneumonia in the United States. *Am J Infect Control*; 2017. [pii: S01966553(17)31056-8]
10. Liu L, Johnson HL, Cousens S, Perin J, Scott S, Lawn JE, et al. Global, regional and national causes of child mortality: An updated systematic analysis for 2010 with time trends since 2000. *Lancet.* 2012;379(9832):2151–61.
11. Jain S, Williams DJ, Arnold SR, Ampofo K, Bramley AM, Reed C, et al. Community-acquired pneumonia requiring hospitalization among US children. *N Engl J Med.* 2015;372(9):835–845.
12. Swati M. Gadappa, Manas Kumar Behera. *Int J Contemp Pediatr.* 2018;5(6):2098-2102.
13. Liliana Cieza-Yamunaqué, Edgar Coila-Paricahua. Pneumonia associated with mechanical ventilation in the pediatric intensive care unit of a tertiary hospital, 2015-2018. *Rev. Fac. Med. Hum.* 2019;19(3):19-26.
14. Russ CD, Koch IF, Laurso DT, 'Sha R, Suthra C, Mackintosh L. *Journal of Hospital Infection.* 2016;92:273e279.
15. Ibrahim ShS. Hospital acquired sepsis in Tanta Pediatric Intensive care Unit. M.sc. Thesis, Pediatric, Tanta University, Faculty of Medicine; 2014.
16. El-Bayoumi MA, El-Nady GM, Badr RI. Clinical and microbiological study of nosocomial infection in pediatric intensive care unit (PICU) in Mansoura University Children's Hospital. *Egypt J of Med Microb.* 2006;15:493-503.
17. Arnoni MV, Berezin EN, Martino M. Risk factors for nosocomial bloodstream infection caused by multidrug resistance gram-negative bacilli in pediatrics. *Baz J Infec Dis.* 2007;11(2):267-271.
18. Gupta A, Kapil A, Lodha R, Kabra SK, Sood S, Dhawan B, et al. Burden of healthcare-associated infections in a pediatric intensive care unit of a developing country: A single center experience using active surveillance. *Journal of Hospital Infection.* 2011;78:323-6.
19. Saied T, ElKholy A, Hafez S, Basim H, Wasfy M, El-Shoubary W, et al. Antimicrobial resistance in pathogens causing nosocomial bloodstream infections in university hospitals in Egypt. *Am J Infect Control.* 2011;39:61-65.
20. Becerra MR, Tanta Lean JA, Suarez VJ, Alvarado MC, Candela JL, Urica FC. Epidemiologic surveillance of nosocomial infection in a pediatric intensive care unit of a developing country. *BMC Pediatrics.* 2010;10-66.
21. Garb JL, Brown RB, Garb JR, et al. Differences in etiology of pneumonia in nursing home and community patients. *JAMA.* 1978;240:2169-72.
22. Mohamed Eida, Mohamed Nasser, Nermine El-Maraghy, Khaled Azab. *Egyptian Journal of Chest Diseases and Tuberculosis.* 2015;64:625–31.
23. Mervat Gamal Eldin Mansour, Sherin Bendary. *The Egyptian Journal of Medical Human Genetics.* 2012;13:99–105.
24. Nicolas Garin, Christophe Marti, Max Scheffler, Jérôme Stirnemann, Virginie Prendki. *Current Opinion in Pulmonary Medicine.* 2019;25(3):242–8.
25. Luna CM, Blanzaco D, Niederman MS, et al. Resolution of ventilator associated pneumonia: Prospective evaluation of the clinical pulmonary infection score as an early clinical predictor of outcome. *Crit Care Med.* 2003;31:676–682.

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